FIG. **6**: Potential relevance of changes in expression of each of the five confirmed proteins detectable in blood of PBBI or MCAo injured rats (i.e. STAT3, Tau, 14-3-3H, p43/EMAP-II, and PKA_{RIIE}) with injury-related mechanisms of apoptosis, neuroinflammation, and intracellular signaling. Utilization of this type of information in combination with other known protein markers (i.e. spectrin break-down products (SBDP), S-100E, myelin break down products (MBDP), myelin basic protein (MBP), and glial acidic fibrillary protein (GFAP)) could have mechanistic (i.e. biomarkers) or pathological (i.e. surrogate markers) relevance to the differential diagnosis/prognosis and treatment of brain-injured patients.

FIG. **7**: Digital image of preliminary Western Blotting results indicating the time course expression profile of p43/EMAP-II in rat brain at different time points post MCAo/PBBI. Blots are to be read from left to right in pairs. For MCAo/MCAo sham, it can be seen in each of the pairs, the p43/EMAP-II expression in MCAo is less than the sham at each of 6 h, 24 h, 48 h and 72 hours. For PBBI/PBBI sham, it can be seen in each of the pairs, the p43/EMAP-II expression in PBBI is greater than the sham at each of 6 h, 24 h, 48 h and 72 hours.

FIG. **8**: Digital image of Western Blots indicating p43/EMAP-II protein expression in different types of tissues. It can be seen that EMAP-II is primarily expressed in brain tissue with minimal expression in other types of tissue such as testes.

FIG. **9**: Conceptual drawing of a rat brain showing method for inducing MCAo.

FIG. **10**: Conceptual drawing of a rat brain showing method used for inducing PBBI.

Human Data:

[0059] Using sandwich ELISA specific to human EMAP-II, CSF (20 μL) and serum (50 μL) samples from a human traumatic brain injury patient (at different post-injury time points) and pooled uninjured controls were analyzed. It was found that EMAP-II levels were elevated in CSF (a) and serum (b) in human TBI samples, when compared to human controls. Table 3 shows the results. In addition, EMAPII levels in pooled CSF and serum samples from TBI patients at some time points were also elevated when compared to those in pooled uninjured controls.

TABLE 3

		[EMAPII] ng/mL
	Human CSF samples	
(a)	pooled Control	0.000
	pooled TBI/0 h	0.000
	pooled TBI/24 h	0.000
	pooled TBI/48 h	0.000
	pooled TBI/72 hr	0.306
	pooled TBI/96 hr	0.000
	#16/0 hr	0.334
	#16/12 h	1.077
	#16/24 h	1.471
	#16/48 h	0.687
	#16/72 h	0.509
	#16/120 h	0.008
	#16/168 h	0.000
	#34/24 h	0.300
	#34/48 h	0.000
	#34/72 h	0.002
	#34/168 h	0.000

TABLE 3-continued

		[EMAPII] ng/mL
	Human serum samples	
(b)	pooled Control	0.059
	pooled TBI/0 h	0.133
	pooled TBI/48 h	0.043
	pooled TBI/72 hr	0.163
	pooled TBI/96 h	0.000
	#16/0 h	0.902
	#16/12 h	0.160
	#16/120 h	0.178
	#16/72 h	0.064
	#16/168 h	0.240
	#34/0 h	0.045
	#34/12 hr	0.204

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